



# UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE  
United States Patent and Trademark Office  
Address: COMMISSIONER FOR PATENTS  
P.O. Box 1450  
Alexandria, Virginia 22313-1450  
[www.uspto.gov](http://www.uspto.gov)

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/623,891	07/21/2003	Sanjay M. Reddy	0167.03	6885
25712	7590	09/08/2006	EXAMINER	
USDA-ARS-OFFICE OF TECHNOLOGY TRANSFER NATIONAL CTR FOR AGRICULTURAL UTILIZATION RESEARCH 1815 N. UNIVERSITY STREET PEORIA, IL 61604				SALVOZA, M FRANCO G
ART UNIT		PAPER NUMBER		
		1648		

DATE MAILED: 09/08/2006

Please find below and/or attached an Office communication concerning this application or proceeding.



UNITED STATES PATENT AND TRADEMARK OFFICE

Commissioner for Patents  
United States Patent and Trademark Office  
P.O. Box 1450  
Alexandria, VA 22313-1450  
[www.uspto.gov](http://www.uspto.gov)

**BEFORE THE BOARD OF PATENT APPEALS  
AND INTERFERENCES**

Application Number: 10/623,891  
Filing Date: July 21, 2003  
Appellant(s): REDDY ET AL.

**MAILED**  
**SEP 08 2006**  
**GROUP 1600**

---

Reddy et al.  
For Appellant

**EXAMINER'S ANSWER**

This is in response to the appeal brief filed May 12, 2005 appealing from the Office action mailed December 2, 2004

**(2) Related Appeals and Interferences**

The examiner is not aware of any related appeals, interferences, or judicial proceedings which will directly affect or be directly affected by or have a bearing on the Board's decision in the pending appeal.

**(3) Status of Claims**

The statement of the status of claims contained in the brief is correct.

**(4) Status of Amendments After Final**

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

**(5) Summary of Claimed Subject Matter**

The appellant's statement of the grounds of rejection to be reviewed on appeal is substantially correct. The changes are as follows: Appellants argue in their brief that they believe that the increased rate of replication is the result of the insertion of the reticuloendotheliosis virus LTR unto the genome of the Marek's disease virus upstream of the ICP4 gene. However, this is not a claimed feature of the invention, which is only drawn to the recombinant Marek's disease virus strain CVI988 transformed with a foreign DNA construct which includes a long terminal repeat sequence (LTR) of a reticuloendotheliosis virus.

**(6) Grounds of Rejection to be Reviewed on Appeal**

The appellant's statement of the grounds of rejection to be reviewed on appeal is correct.

**(7) Claims Appendix**

The copy of the appealed claims contained in the Appendix to the brief is correct.

**(8) Evidence Relied Upon**

Jones et al., "Retroviral Insertional Activation in a Herpesvirus: Transcriptional Activation of Us Genes by an Integrated Long Terminal Repeat in a Marek's Disease Virus Clone," Journal of Virology, Vol. 70, No. 4 (April 1996), pp. 2460-2467.

Witter, R.L., Lee, L.F. and Fadley. A.M., "Characteristics of CVI988/Rispens and R2/23, Two Prototype Vaccine Strains of Serotype 1 Marek's Disease Virus," Avian Diseases (1995) 39: pp. 269-284.

R. L. Witter. A Deshan Li, Dan Jones, L. F. Lee, and H.-J. Kung, "Retroviral Insertional Mutagenesis of a Herpesvirus: A Marek's Disease Virus Mutant Attenuated for Oncogenicity But Not for Immunosuppression or In Vivo Replication," Avian Diseases Vol. 41 (1997), pp. 407-421.

**(9) Grounds of Rejection**

The following ground(s) of rejection are applicable to the appealed claims:

***Claim Rejections - 35 USC § 103***

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

**Claim group 1, claims 1, 2, 5-9 and 12-15**

Claims 1, 2, 5-9 and 12-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over Witter et al. (1997), Witter et al. (1995), and Jones et al. (1996).

Appellant claims a recombinant Marek's disease virus (MDV) CVI988/X that is stably transformed with the long terminal repeat sequence (LTR) of reticuloendothelial virus (REV) where the recombinant virus is effective to elicit an immune response in a chicken to Marek's disease virus without causing a significant degree of pathogenicity in the chicken. Appellant indicates that MDV CVI988/X includes, but is not limited to, MDV CV1988 or any of its clones, including CVI988/Rispens (page 13 of the specification).

Witter et al. (1997) *Avian Diseases*, 41:407-421 discloses a recombinant MDV, referred to as RM1, based upon the JM/102W strain of MDV, which was stably transformed with the LTR of REV (page 408) (See also Jones et al, *J. Virol.* (1996) p. 2460-67 which describes more fully the creation of RM1). Witter et al. reported that RM1 was effective to elicit an immune response in chickens (page 413) and that the response was highly protective upon challenge (Table 7 on page 418), greatly exceeding that of the attenuated serotype 1 vaccine strains

CVI988 and JM/102W (upon which it was based). Witter et al. reports that JM/102W (page 48), like other attenuated serotype 1 MDVS, replicates poorly in vivo (page 418). In contrast, RM1 replicated efficiently in vivo. Moreover, RM1, like the attenuated viruses often used in vaccines, did not result in significant oncogenicity (page 416). Witter et al. speculates that the LTR insertion induced the greater in vivo replication, which, in turn, resulted in the increased protection in RM1 vaccinated chickens upon challenge with virulent MDV (page 418). Witter et al. points out that RM1 is not a good candidate for commercial vaccine development due to its ability to cause persistent thymic atrophy and residual oncogenicity, but that it does represent a model for future vaccine development (page 420). In particular, Witter et al. provides that the "selective mutation of key genes (with REV) will prove to be a useful strategy for development of superior serotype 1 vaccines." (See last sentence of text on page 420).

Witter et al. (1997) does not teach using the CV1988 strain of MDV. Additionally, Witter et al.'s RM1 strain caused a significant degree of pathogenicity in the chicken in terms of its ability to cause thymic atrophy.

Witter et al., *Avian Diseases* (1995) 39:269-284 reports on an attenuated serotype 1 MDV vaccine virus known as CVI988/Rispens that does not result in thymic atrophy (page 282). CVI988/Rispens resulted in the best protection when compared with other vaccines (abstract).

One of ordinary skill in the art would have been motivated to substitute the JM/102W strain of MDV as taught by Witter et al (1997) with the CVI988/Rispens as taught by Witter et al (1995) to create a recombinant MDV vaccine because Witter et al (1997) states that selective mutation with REV may be an advantageous strategy for the development of superior serotype 1 MDV vaccines. Additionally, the incorporation of LTR-REV will increase viral replication

capacity (see the first paragraph of “Results” on p. 275 of Witter et al. (1995) stating that the replication capacity of CVI988/Rispens was inferior to another strain). One of ordinary skill in the art would have had a reasonable expectation of success in producing a recombinant virus through the transformation of CVI988/Rispens with REV to yield an MDV vaccine effective to elicit an immune response in a chicken to MDV without causing significant pathogenicity because MDV-REV transformants have been previously shown to be effective at eliciting an immune response while CVI988/Rispens has been shown to have the characteristic of not inducing thymic atrophy. Additionally, one would have had a reasonable expectation of success for increasing the growth rate by incorporation of LTR-REV into CVI988/Rispens since LTR-REV increases the growth rate in another recombinant Marek’s Disease Vaccine strain (see Witter et al. (1997)). One would have been further motivated to substitute the JM/102W strain of Witter et al. (1997) with the CVI988 of Witter et al. (1995) because CVI988 is more attenuated since it does not cause thymic atrophy (see p. 282). Therefore, the invention as a whole would have been *prima facie* obvious to one of ordinary skill in the art at the time of the invention.

As to claim 2 the specification indicates on page 13 that RM1 was used as the source of the REV LTR used in the present MDV recombinant. Therefore, it is reasonable to conclude that by applying the teachings of Witter et al (1997) over Witter et al (1995), as outlined above, one would produce a viral agent wherein said LTR comprises Sequence ID No. 2. Furthermore, Jones et al provides the details of the MDV RM1 vaccine virus used by Witter et al (1997) and is explicitly referenced by Witter throughout page 408. The REV LTR sequence disclosed by Jones et al in Fig. 1 on page 2461 is identical to Sequence ID No. 2 as provided by Appellant.

As to claims 5, 6, 7, 8, 12, 13, 14 and 15 it is reasonable to conclude that by applying the teachings of Witter et al (1997) over Witter et al (1995), as outlined above, one would produce a viable (see claims 6 and 13) MDV viral agent having all of the identifying characteristics of ATCC P7-A-9495 (see claims 5 and 12). One would also produce a vaccine comprising the viral agent in an amount effective to elicit a protective immune response in a chicken to Marek's disease virus and a pharmaceutically acceptable carrier or diluent (see claim 8). Furthermore, cell-association is widely regarded as a property of many enveloped viruses including those in the family Herpesviridae (see claim 7). For instance, Jones et al, *J. Virol.* (Apr. 1996) p. 2460-67, indicates on page 2460, first paragraph of the Materials and Methods section, that MDV is typically cell-associated when propagated in vitro. Thus, one would expect any MDV virus to be cell-associated as an inherent property of the virus and irrespective of the REV LTR insert. Claim 14, like claim 7, concerns the cell-associated nature of the virus vaccine, and claim 15 claims a "method for making a viral agent effective for protecting a chicken against Marek's disease comprising transforming a Marek's disease virus strain CV1988 with a foreign DNA construct which comprises a long terminal repeat sequence of a reticuloendotheliosis virus." This claim stands rejected under the art of record, Witter et al (1997) over Witter et al (1995) as applied to claims 1, 2, 5, 6, 9, and 13 the previous Office Action dated June 16, 2004.

Claim group 2, claims 3 and 10

Claims 3 and 10 are generally directed to a viral agent; product claims. The claims indicate "said long terminal repeats sequence comprises a *Pac I* excised DNA segment from a Marek's disease virus..." It is the Examiner's understanding that the long terminal repeat sequence is excised from a larger nucleic acid via *Pac I* restriction

endonuclease digestion. Thus, one would be left with a linear nucleic acid with the partial *Pac I* palindrome at the extreme ends of the sequence. Paragraph [0055] of the specification indicates that this sequence is inserted into B40-Pac, which is then digested with *Not I*. It is not clear whether or not the *Not I* excised fragment that is to be recombined still has the *Pac I* site and, more importantly, whether the *Pac I* site is maintained during recombination. This *Not I* digested nucleic acid would then be used to transform a virus via recombination. The sequence that would be integrated would be internal to either extreme end. The point of this is that the identity of the particular restriction site would appear to be irrelevant since the site would be lost during recombination. The long terminal repeat is not adding/delivering a *Pac I* site to the virus to which it transforms, though this seems to be what the claim is suggesting. The bottom line is that the process by which the LTR was excised does not impart any distinctive structural characteristics to the final product. That a *Pac I* site “would most likely be present in the recipient CVI988 MDV strain (or any other CVI988 strain) (into which the LTR subsequently recombines) because the herpesvirus genome is highly conserved” is a tangential matter and does not resolve the problems of claims 3 and 10 as outlined above.

#### **(10) Response to Argument**

##### Claim group 1, claims 1, 2, 5-9 and 12-15

Claims 1, 2, 5-9, and 12-15 have been rejected under 35 U.S.C. 103(a) as being unpatentable over Witter et al. (1997) in view of Witter et al. (1995), and further in view of Jones et al. (1995).

Appellant argues in its brief in regards to Witter et al. that although the RM1 strain was shown to provide a level of protection similar or superior to that of CVI988, it was associated with residual pathogenicity, caused thymic atrophy in treated birds, and the reason why the RM1 strain possessed these properties "cannot definitively be established" and speculated that there were several possible mechanisms for the RM1 strain activity.

Appellant's arguments are unpersuasive, since they are taken out of context and unsupported by the teachings. Witter et al., discusses the benefits of the RM1 strain (p. 418), even though the mechanism of the benefits cannot be explained.

Appellant further argues that Witter et al. concluded that the RM1 may provide a model for future vaccines, "if" the superior protection by RM1 clones derives from the selective attenuation of oncogenicity without influence on in vivo replication of other properties, then "perhaps selective mutation of key genes" will prove useful for developing superior vaccines (pp. 419-420).

Appellant's arguments are unpersuasive, since they are also taken out of context and unsupported by the teachings. Witter et al., discusses these possibilities (p. 418) in the context of unknown mechanisms associated with LTR insertion.

Appellant's arguments are also based upon the assertion that "a practitioner of ordinary skill in the art would still have no motivation to combine the references as suggested, nor would they have any reasonable expectation of success" based upon the combined teachings of Witter et al. (1997) in view of Witter et al. (1995), or further in view of Jones et al. (1996) in regards to cancelled claims 4 and 11. Appellant has further asserted that Examiner's reasoning is based upon an "obvious to try" rationale.

As stated above, this is not persuasive.

### 1. No Motivation

First, the combined teachings of Witter et al. (1997) in view of Witter et al. (1995) would motivate one of ordinary skill in the art. In summary, Witter et al (1997) teaches a viral agent comprising recombinant Marek's disease virus (RM1) stably transformed with a foreign DNA construct which comprises a long terminal repeat sequence of reticuloendotheliosis virus, wherein said viral agent is effective to elicit an immune response in a chicken to Marek's disease virus, and further wherein said long terminal repeat sequence is inserted upstream of the ICP4 gene of said Marek's disease virus. Witter et al. (1997) does not teach the strain CVI/988X nor does he teach a viral agent that does not cause a significant degree of pathogenicity, as claimed in claim 1, though the reference does make numerous efficacy comparisons to CVI/988 and substantially recognizes the problem of the pathogenicity of the vaccine. In the second to last sentence, Witter et al (1997) indicates that the principal problem with the use of RM1 as a vaccine was the issue of thymic atrophy (i.e. it caused a significant degree of pathogenicity).

In closing, Witter et al. (1997) states that selective mutation (with REV) may be an advantageous strategy for the development of superior serotype 1 MDV vaccines. This is both motivation and a suggestion. The suggestion would be to find a means to overcome the issue of thymic atrophy.

Witter et al. also reports that this RM1 strain provided superior protection to the CVI988 strain though the parent strain from which RM1 was derived provided inferior

protection, which is relevant to the present application. (See also table 7) Witter et al. reports that the next best vaccine in terms of protective ability to the RM1 strain was CVI988/Rispens vaccine (pg. 419, column 2). Witter et al. (1997) concludes by pointing out that RM1 is not a likely candidate for commercial vaccine development due to its tendency to cause persistent thymic atrophy. (See second to last sentence of Witter et al. (1997) straddling pgs. 419-420.) As noted above, this is a clear suggestion to adopt a strain with a reduced potential for thymic atrophy. Given the protection shown in Witter et al. (1997) one would be motivated to create similar vaccine constructs using LTR inserted into MDV.

Unlike JM/102W into which Witter et al. (1997) inserted the LTR, Witter et al. (1995) report that CVI988/Rispens does not induce thymic atrophy. Moreover CVI988 was the vaccine strain that Witter et al (1997) reported as second to only RM1 in protective efficacy. Therefore, motivation exists to combine the references.

## 2. No Reasonable Expectation of Success

Second, one would have a reasonable expectation of success. The prior art can be modified or combined to reject claims as *prima facie* obvious as long as there is a reasonable expectation of success. *In re Merck & Co., Inc.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986) Obviousness does not require absolute predictability, however, at least some degree of predictability is required. Evidence showing there was no reasonable expectation of success may support a conclusion of nonobviousness. *In re Rinehart*, 531 F.2d 1048, 189 USPQ 143 (CCPA 1976) Whether an art is predictable or whether the proposed modification or combination of the prior art has a reasonable expectation of success is determined at the time the invention was made. *Ex parte Erlich*, 3 USPQ2d 1011 (Bd. Pat. App. & Inter. 1986)

The techniques involved in creating a recombinant MDV vaccine harboring the LTR of REV were well established at the time of Appellant's invention and these techniques are taught in a number of the references cited by Appellant including Witter et al. (1997). Therefore one would have a reasonable expectation of success in making the viral agent claimed by Appellant. As noted above, this does not require absolute predictability. Appellant has not provided evidence that shows that, at the time of Appellant's invention, there would be no reasonable expectation of success. Appellant has submitted Parcells et al (2004).

Appellant points to the Parcells et al (2004) abstract as evidence that one would have no reasonable expectation of success in creating the claimed invention as suggested by the combining of the two Witter references. This appears erroneous for a number of reasons. First, reasonable expectation of success must be determined at the time of Appellant's invention. As for the Parcells et al. abstract, this post-dates Appellant's invention by at least one year. One skilled in the art could not be daunted by unknown obstacles although the subsequent publications might be relevant to show that the obstacles actually applied to the specific problems facing the inventor. *Velander v. Garner*, 68 USPQ 2d 1769, 1783 (CAFC 2003)

Second, Appellant has suggested that Parcells et al. represents failure because the resulting transformants containing the inserted LTRS did not exhibit enhanced replication. However, enhanced replication is not a claimed feature of Appellant's invention. Consequently, it appears at first blush that Parcells et al. succeeded in doing exactly what Appellant is claiming, though they may have failed in that the insert was downstream of the ICP4 gene? If so, this might further explain the reduced replication relative to Witter et al (1997).

Finally, that Parcells et al. attempted to do what is taught by the combined Witter

references, and Appellant's invention, seems to bolster the notion that one would be motivated to do this and have a reasonable expectation of success in so doing. Otherwise Parcells et al. would not have attempted it. It is noted that the Witter et al. (1997) publication has authors who are also inventors on the present application. It has been outlined above how Witter et al. (1997) in view of Witter et al. (1995) provides the motivation and suggestion in support of the present rejection. (See especially final line of Witter et al (1997)). It is noted that Appellants assert that one would have no reasonable expectation of success based on the statements in Witter et al. (1997). This is not found persuasive.

Appellant also notes that Parcells clearly illustrates the unpredictability of repeating the process of Witter '97 with other Marek's disease virus strains, and that Parcells et al. is not available as prior art under any section of 35 U.S.C. 102/103 and cannot be relied upon to provide motivation for combining the references as suggested.

Appellant's arguments in regards to the Parcells et al. reference are unpersuasive since Parcells et al. is not cited specifically as prior art to provide motivation in the rejection, rather as proof that that one would be motivated to do it and have a reasonable expectation of success. Second, Parcells et al. taught only that the insertion of the LTR sequence from RM1 at "*the identical location within the IRs* (repeat flanking the short region)" (emphasis added) may not have conferred enhanced replication, but that "the inability of the RM1-like LTR insertion in a CVI-988 background to confer enhanced replication *in vivo* may be due to slight differences of the insert sequence (Lox site), or insertion in only one of the two inverted repeats. Mutations other than the LTR insertion *within the IRs/TRs* (repeats flanking the short region) may also contribute to the enhanced replication of RM1" (emphasis added). In other words, Parcells et al.

offers that they succeeded in doing what Appellant is claiming, though insertions in certain locations may contribute more than insertions in others.

### 3. Obvious to Try

As a final matter, Appellant has asserted that the Examiner's rejection was based merely on an "obvious to try" rationale. As provided in *In re Merck & Co., Inc.*, 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986):

"[O]bvious to try is not the standard of 35 U.S.C. § 103." *In re Antonie*, 559 F.2d 618, 620, 195 USPQ 6, 8 (CCPA 1977) (emphasis omitted). Rather, the test is whether the references, taken as a whole, would have suggested appellant's invention to one of ordinary skill in the medicinal chemical arts at the time the invention was made. *In re Simon*, 461 F.2d 1387, 1390, 174 USPQ 1 14, 1 16 (CCPA 1972). Obviousness does not require absolute predictability. *In re Lamberti*, 545 F.2d 747, 750, 192 USPQ 278, 280 (CCPA 1976). Only a reasonable expectation that the beneficial result will be achieved is necessary to show obviousness. *In re Longi*, 759 F.2d 887, 897, 225 USPQ 645, 651 (Fed. Cir. 1985).

Appellant is correct to the extent that obvious to try is not the proper standard. Nevertheless, the rejection was based on more than merely an obvious to try rationale. As outlined above, one would have had a reasonable expectation that a beneficial result would be achieved. Moreover, the obvious to try rationale is not on point in the present instance. The following is an excerpt from MPEP 2145:

"The admonition that 'obvious to try' is not the standard under § 103 has been directed mainly at two kinds of error. In some cases, what would have been 'obvious to try' would have been to vary all parameters or try each of numerous possible choices until one possibly arrived at a successful result, where the prior art gave either no indication of which parameters were critical or no direction as to which of many possible choices is likely to be successful... . In others, what was 'obvious to try' was to explore a new technology or general approach that seemed to be a promising field of experimentation where the prior art gave only general guidance as to the particular form of the claimed invention or how to achieve it." *In re O'Farrell*, 853 F.2d 894, 903, 7 USPQ2d 1673, 1681 (Fed. Cir. 1988)

No suggestion was made to vary a multitude of parameters nor was the suggestion one of the exploration of new technology or general approaches. Instead, the motivation and suggestion was quite specific. Therefore, Appellant's arguments are not found persuasive.

Appellant also notes that Jones et al. and Witter et al. (1997) only disclose that a “potential” ICP4 promoter is upstream from the LTR insertion site and that “it is conceivable” that the ICP4 transactivator may be significant, does not provide a reasonable expectation of success, and, at best only suggests that it would be reasonable to try insertion of the LTRs at that location and moreover, the reference provides no guidance how the site of insertion could be controlled to ensure insertion of an LTR at that location.

Jones et al. provides the details of the MDV RM1 vaccine virus used by Witter et al. (1997) and is explicitly referenced by Witter throughout page 408. In particular, Jones teaches that the insertion of the LTR is in the vicinity of the MDV ICP4 gene (see pg. 2466; col. 1, last full sentence). Jones states, “Since a potential promoter for the long form of the MDV ICP4 gene is located 100 to 200 bp upstream from the LTR insertion site, it is conceivable that the ICP4 transactivator may also be associated with the novel phenotype of RM1.” The quote is relevant for two reasons. First, where the promoter for ICP4 is upstream of the LTR, it is reasonable to conclude that the insertion of the LTR was in between the ICP4 promoter and the ICP4 gene, which would place the LTR upstream of the ICP4 gene upstream as specified in amended claims 1 and 9. This conclusion is bolstered by the observation in Jones et al. that integration patterns are clustered and region-specific. (See pg. 2465, column 1) Second, Jones et al. correlates the desired phenotypic change with the locality of the insertion of the LTR to create a reasonable expectation of success. As an academic matter, given that Witter et al (1997) references Jones et al for support on construction of RM1, all of the limitations are properly attributable to Witter in view of Witter without resort to Jones except for explanation.

Appellants indicate on page 11 of the Non-Final Rejection response (final line) that the

increased rate of replication is the result of the insertion of the reticuloendotheliosis virus LTR into the genome of the Marek's disease virus upstream of the ICP4 gene. Further, it is stated, "This is not disclosed or suggested in the prior art." In fact, this appears to be exactly the thing that is taught in the suggested in Jones et al./ Witter et al (1997) sequence of papers dealing with the RM1 strain containing the LTR.

Appellants state on page 14 of the Non-Final Rejection response, "It is only by virtue of Appellants' invention that the point of insertion can be controlled and LTRs can be introduced into other Marek's disease viruses to produce effective vaccine." It is the Examiner's understanding that the LTR enters the MDV genome by homologous recombination. As mentioned above, Jones et al. teaches that integration patterns are clustered and region specific. (See also Jones et al. PNAS (1993) cited by Appellant as AT1) It is not evident how Appellant's invention controls the point of insertion to any greater extent than that seen in Jones et al. (1995) Unlike the addition of a nucleic acid sequence via restriction digest and ligation, homologous recombination is somewhat random.

Claim group 2, claims 3 and 10

Appellants also believe that dependent claims 3 and 10 further differentiate over the prior art of record for reciting process limitations describing that the long terminal repeat of the independent claims "comprises a Pac I excised DNA segment from Marek's disease virus strain ATCC PTA-4945," which is not disclosed or suggested in the prior art of record, and even if it did, it provides no teaching to do so.

The rejection is maintained because the particular process step involving the Pac I excision does not impart distinctive characteristics to the final product because the Pac I site is external to the fragment that joins to the virus during the recombination event. Therefore, it fails to define the product over the combined teachings of the cited prior art.

**(11) Related Proceeding(s) Appendix**

No decision rendered by a court or the Board is identified by the examiner in the Related Appeals and Interferences section of this examiner's answer.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

  
M. Franco Salvoza

Conferees:

Bruce Campell



BRUCE R. CAMPELL, PH.D  
SUPERVISORY PATENT EXAMINER  
TECHNOLOGY CENTER 1600

Jeffrey Siew

  
JEFFREY SIEW  
SUPERVISORY PATENT EXAMINER